

Intertooth Patterns of Hypoplasia Expression: Implications for Childhood Health in the Classic Maya Collapse

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ABSTRACT Enamel hypoplasias, which record interacting stresses of nutrition and illness during the period of tooth formation, are a key tool in the study of childhood health in prehistory. But interpretation of the age of peak morbidity is complicated by differences in susceptibility to stress both between tooth positions and within a single tooth. Here, hypoplasias are used to evaluate the prevailing ecological model for the collapse of Classic Period Lowland Maya civilization, circa AD 900. Hypoplasias were recorded in the full dentition of 160 adult skeletons from six archaeological sites in the Pasión River region of Guatemala. Instead of constructing a composite scale of stress experience, teeth are considered separately by position in the analysis. No statistical differences are found in the proportion of teeth affected by hypoplasia between "Early," Late Classic, and Terminal Classic Periods for anterior teeth considered to be most susceptible to stress, indicating stability in the overall stress loads affecting children of the three chronological periods. However, hypoplasia trends in posterior teeth may imply a change in the ontogenetic timing of more severe stress episodes during the final occupation and perhaps herald a shift in child-care practices. These results provide little support for the ecological model of collapse but do call to attention the potential of posterior teeth to reveal subtle changes in childhood morbidity when considered individually. *Am J Phys Anthropol* 102:233-247, 1997 ©1997 Wiley-Liss, Inc.

Enamel hypoplasias are a common focus of bioarchaeological research that addresses health transitions in prehistory. These dental defects are particularly useful because they record nutritional and health stresses during childhood, a crucial period of life. Childhood health is directly linked to the demographic structure of a society and to population processes at large. Because sub-adult remains are often underrepresented in archaeological skeletal series, hypoplasias provide a unique record of childhood stress experience observable in adult skeletons.

Enamel is laid down first as a protein matrix secreted by ameloblasts in the dental papilla. Hydroxyapatite crystals then form

in this matrix along flow lines generated by ameloblastic secretions. The resulting enamel mineral has a prismatic structure; each prism records the paths taken by four adjacent ameloblasts over their secretory lives (Osborn, 1973). Nutritional or disease stresses that disrupt the ameloblastic secretory rate result in reduced enamel thickness. As amelogenesis commences at the tooth cusp and progresses cervically, this disruption is permanently registered as a ring or circumferential groove of deficient or

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pitted enamel, a hypoplasia. Since the chronology of dental development is well understood, the age at which the stress episode occurred can be estimated from the position of the hypoplasia on the tooth crown with some accuracy (Goodman and Rose, 1990; Suckling, 1989). Accordingly, hypoplasias are enticing tools for reconstructing health patterns during childhood.

The ability to pinpoint the timing of stress episodes during dental growth makes hypoplasias a key approach to study of weaning practices in prehistory. Peak ages of hypoplasia occurrence typically are interpreted as age of weaning, often between 2 and 4 years (Cook and Buikstra, 1979; Corruccini et al., 1985; Moggi-Cecchi et al., 1994; Rathbun, 1987; Saul, 1972). This hypothesis is often assumed to be true or is implied but has not been free from criticism (Blakey et al., 1994; Danforth, 1989). Since weaning is a critical period for child survival, documentation of prehistoric weaning practices and their demographic implications is fundamental to bioarchaeological investigation.

Studies of the chronological distribution of hypoplasias often attempt to construct a composite score of stress experience using multiple teeth or the complete dentition (Blakey and Armelagos, 1985; Blakey et al., 1994; Cook and Buikstra, 1979; Corruccini et al., 1985; Goodman and Armelagos, 1988; Goodman et al., 1980; Moggi-Cecchi et al., 1994; Whittington, 1992). This method allows study of a longer period of development than registered in a single tooth. Typically defect locations are converted into developmental ages, and then 6 month intervals of growth are scored as positive or negative for growth disruption, as observed on a minimum number of teeth scorable for that interval (Goodman et al., 1980). This composite scale is used to identify the peak age of hypoplastic stress. However, such attempts are complicated by two issues: for each tooth hypoplastic sensitivity is conditioned by aspects of crown geometry, and different tooth positions are not equally susceptible to stress (Goodman and Armelagos, 1985a,b; Condon and Rose, 1992).

Hypoplasias are most abundant in the middle third of the enamel of each tooth. Not simply governed by the timing of stress

episodes, this distribution appears to be partly influenced by crown geometry. Goodman and Armelagos (1985a) suggest that deficiencies of enamel depth may be more visible in this region due to the near perpendicular orientation of enamel prisms, in contrast to the more oblique orientation of cuspal prisms. Hypoplasias visible on the crown surface are therefore biased toward the middle period of amelogenesis for each tooth. For this reason, peak occurrence of hypoplasia on a given tooth do not directly mirror the timing of peak stress experience. Moreover, hypoplastic age distributions cannot be directly compared among different tooth positions. Accordingly, some recent studies have not attempted to link teeth into a composite scale (Lanphear, 1990; McKee and Lunz, 1990; Storey, 1992a,b).

Goodman and Armelagos (1985a,b) also observed that hypoplasias are most abundant on maxillary incisors and mandibular canines, a distribution confirmed in many subsequent studies. This finding indicates that these teeth are more susceptible to developmental arrest than are teeth that rarely show hypoplasias. They hypothesize that the polar teeth of developmental fields are more susceptible to hypoplasia because their development is under stronger genetic control. Goodman and Rose (1990) propose that nutritional and disease stress episodes produce hypoplasias only when the magnitude of ameloblast disruption reaches a critical threshold level for a given tooth. Ameloblasts producing posterior and nonpolar teeth are less susceptible than those of anterior teeth, which only record stressors of greater severity. Condon and Rose (1992) further suggest that differences in susceptibility may be related to tooth-specific variation in the rate of enamel deposition, with more slowly deposited enamel more prone to developmental arrest.

Because amelogenesis of anterior teeth (incisors and canines) is more easily disrupted, these teeth appear to record the majority of stress episodes experienced by a child. That is, stress episodes producing defects on posterior teeth are generally also recorded coevally on anterior teeth forming at that time. Goodman and Armelagos (1985b) recommend that hypoplasia studies

concentrate on these sensitive teeth. Many subsequent studies have focused only on incisors and canines, thereby avoiding possible underenumeration of defects on posterior teeth (Goodman et al., 1987, 1991; Hodges, 1987; Hutchinson and Larsen, 1988; Lanphear, 1990; Van Gerven et al., 1990).

In this paper, I examine hypoplasia frequencies in the full dentition. Rather than attempting to control for intertooth susceptibility differences, I consider each tooth independently. I explore the possibility that intertooth differences in susceptibility to developmental arrest can be exploited to provide a more detailed reconstruction of childhood health patterns.

THE ARCHAEOLOGICAL PROBLEM

The collapse of political systems and the abandonment of large Maya cities in the southern part of the Maya Lowlands near AD 900 is a recurrent focus of anthropological inquiry. Explanations put forth to explain the failure of Maya civilization range from class conflict (Thompson, 1970), warfare (Demarest, 1992, 1996), trade (Freidel, 1986; Webb, 1973), and ideology (Puleston, 1979) through environmental degradation (Culbert, 1988; Sanders, 1973), climate change (Hodell et al., 1995), crop blights (Brewbaker, 1979), epidemic disease (Shimkin, 1973; Spinden, 1928), and volcanism (MacKie, 1961). Although political factors are now gaining prominence in discussions of the collapse (Culbert, 1991; Demarest, 1996; Fash, 1994; Miller, 1993), ecological arguments are favored by many Mayanists. Speculations about deteriorating childhood health are ubiquitous in the latter arguments.

The humid tropical forest is often seen as a severe challenge to agriculture, in part because the area has been sparsely settled since the demographic transition of the Maya collapse. Prehistoric population is estimated to have reached a critical density during the Late Classic period, straining agricultural systems beyond productive capacity. It is argued that soil erosion, grass invasion, and deforestation removed land from agricultural use, leading to crop shortages, and reduced the availability of wild faunal resources. These factors contributed to an

increased reliance on maize and beans as staple foods, compromising the quality of the diet. Nutritional deficiency was felt most severely by young children and was responsible for elevated childhood morbidity and mortality, a key factor that precipitated demographic collapse at the end of the first millennium AD (Culbert, 1988; Sanders, 1973; Santley et al., 1986; Saul, 1973; Willey and Shimkin, 1973).

In this paper I reexamine the evidence for changes in childhood health over the span of Classic Maya occupation through the prevalence and patterning of enamel hypoplasias in the dentitions of human skeletons from the Pasión region of the southwestern Petén, Guatemala. Early observations on enamel hypoplasias by Saul (1972, 1973) at two of these sites are commonly cited as evidence of inadequate child nutrition in support of the ecological model of collapse (e.g., Santley et al., 1986). These series merit restudy in the context of recent advances in the theory and methodology of hypoplasia research.

MATERIALS AND METHODS

Skeletal series

This study focuses on six sites in the watershed of the Pasión River: Dos Pilas, Aguateca, Tamarindito, Itzán, Seibal, and Altar de Sacrificios (Fig. 1). Dos Pilas, Tamarindito, and Aguateca were excavated by the Vanderbilt University Petexbatún Regional Archaeological Project between 1989 and 1994 (Demarest and Houston, 1989, 1990; Demarest et al., 1991, 1992; Valdés et al., 1993). Yale University undertook excavations at Itzán in 1990 (Johnston, 1994). Seibal and Altar de Sacrificios were excavated by Harvard University projects during the 1960s (Willey, 1973, 1990; Willey and Smith, 1969; Willey et al., 1975).

Due to the ancient Maya practice of sub-floor domestic burial (rather than the use of corporate cemeteries) and the need for extensive architectural excavation in order to recover burials, Maya skeletal series are generally small. Accordingly, bioarchaeological investigation of the Maya is more feasible on a regional than a site-focused scale. A regional level of analysis is also useful to counterbalance biases in archaeological sam-

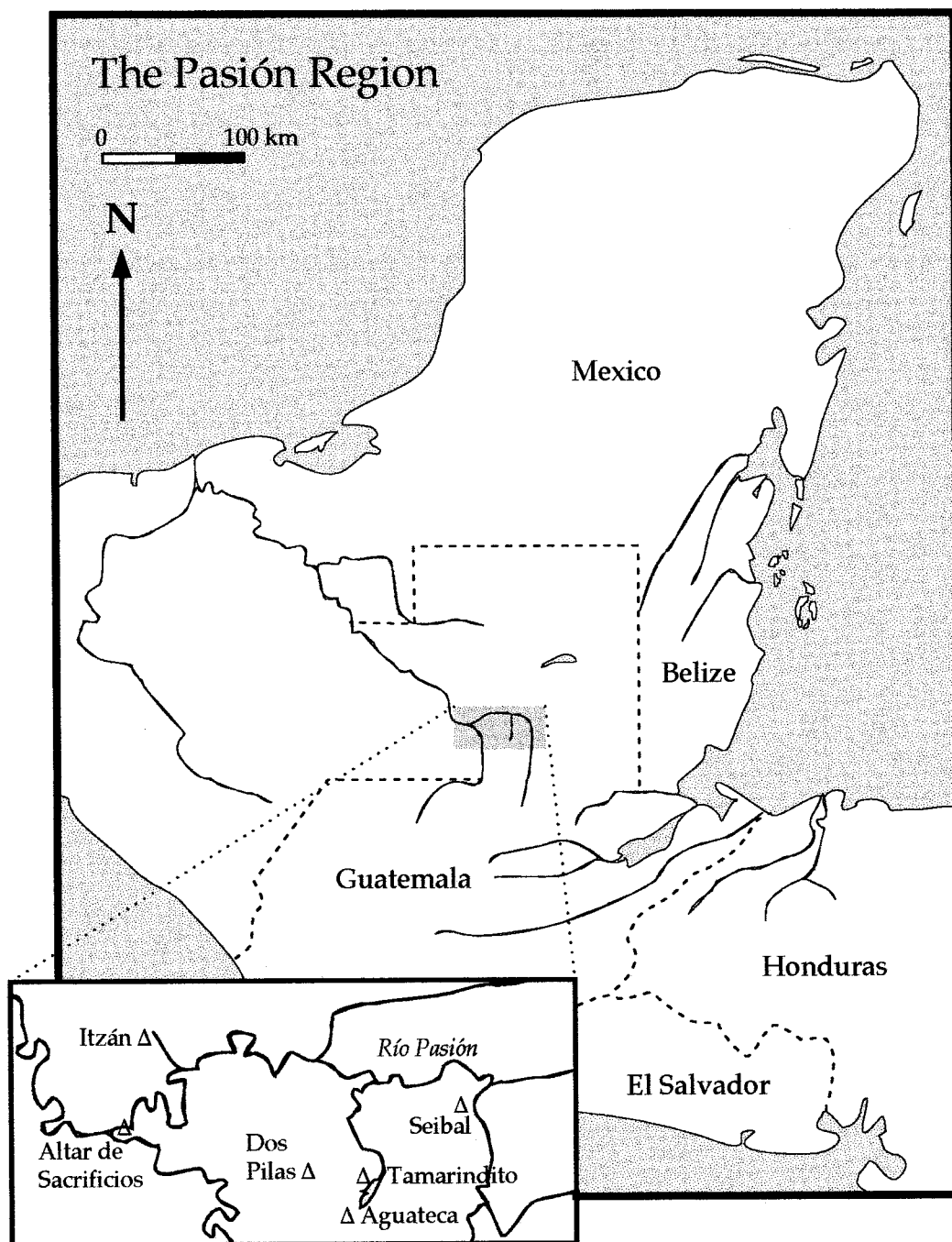


Fig. 1. Map of the Pasión region in the Maya Lowlands.

pling that may result from differing excavation strategies between sites. Regionally specific architectural and ceramic styles distinguish the Pasión from neighboring ar-

eas and are now complemented by textual evidence for intraregional elite interaction (Mathews and Willey, 1991). Although these sites do show some paleodietary distinc-

tions, the isotopic composition of bone collagen is quite similar, with large overlapping ranges at all Pasión sites (Wright, 1994). Across the region, environmental distinctions are not so marked that paleoepidemiological conditions might differ between sites.

The Pasión region is today covered by moist perennial broadleaf forest. Most of the year the region receives heavy rainfall, 2,000 mm annually, punctuated by a brief dry season from March to May. In addition to slow surface rivers, the uplifted karstic terrain is penetrated by a network of caves and underground streams. Ancient agricultural practices included swidden, intensive cultivation of sinkholes and slopeland terracing, but water-table fluctuations are too dramatic for raised or drained fields common in other parts of the Maya Lowlands (Dunning and Beach, 1994). Paleobotanical remains recovered in excavations (Lentz, 1994) and carbon isotopic data on human bone collagen (Wright, 1994) indicate that the Pasión Maya relied heavily on maize agriculture supplemented by cultivated beans, squash, chilis, and a variety of wild fruits. Nitrogen isotopic data do not imply that meat consumption was critically limited (Wright, 1994), as evidenced also by faunal remains of diverse terrestrial and aquatic taxa in domestic middens (Emery, 1991; Pohl, 1985).

The burials studied here are divided among three chronological horizons. The earliest period spans the longest time, from 600 BC to AD 600, and includes both the Preclassic and Early Classic Periods, hereafter referred to as the "Early" period. This era saw the origins of monumental architecture and artistic traditions for which the Maya are famous as well as the emergence of social stratification. While it would be enlightening to examine health transitions within this long span, the rarity of early burials necessitates lumping the Preclassic and Early Classic Periods into a single horizon. "Early" burials have been found only at Altar de Sacrificios and Seibal in the Pasión region. The boundary between the "Early" and Late Classic burial series was drawn between the Veremos and Chixoy phases for Altar de Sacrificios and between the Junco and Tepejilote phases for Seibal (Adams, 1971; Sabloff, 1975).

The Late Classic Period, from AD 600–830, is well studied archaeologically and represented by larger burial series at each of the five sites. Settlement surveys document a dramatic increase in population density throughout the lowlands. At this time, Maya society was highly stratified, and neighboring sites competed in a complex network of both peaceful and warlike relationships. The Late Classic Pasión was dominated by politico-military expansion of the Dos Pilas polity, which held dominion over a number of neighboring sites, including Tamarindito. Toward the end of this period, intensified warfare contributed to the collapse of several sites, especially Dos Pilas and its twin capital, Aguateca (Demarest, 1996; Demarest and Houston, 1989, 1990; Demarest et al., 1991, 1992; Valdés et al., 1993).

The Terminal Classic Period, AD 830–950, represents the final occupation of the region. As attested by hieroglyphic monuments, political authority was maintained only at Seibal but waned there too in the tenth century. Remnant nonelite populations that persisted into the Terminal Classic Period at Altar de Sacrificios and Dos Pilas are represented in the burial series. Unlike Aguateca and Tamarindito, which were fully abandoned during the preceding phase, Itzán shows some Terminal Classic occupation, but no burials were recovered. All of the sites appear to have been depopulated by about AD 950.

The boundary between Late and Terminal Classic Periods is placed at AD 830, when Fine Orange ceramics appear in Pasión assemblages. At Seibal this corresponds to the Tepejilote to Bayal transition (Sabloff, 1975). Equivalent ceramic phases for the Petexbatún sites are the Nacimiento and Sepens phases (Foias, 1993). For Altar de Sacrificios, where the chronology is somewhat problematic, I consider burials dated as late facet Boca phase and/or Jimba to be Terminal Classic. Early facet Boca phase burials (those lacking late facet diagnostic types) and Pasión phase burials were considered to be Late Classic in date. Advocated by Sabloff (1975), this classification brings the ceramic chronology for Altar (Adams, 1971) into line with that of Seibal and the Petexbatún sites.

This study employs the dentitions of 160 adult skeletons. Subadult skeletons are rare

in these series and insufficient for study in their own right. Individuals who died during childhood may show more defects than those who survived to adulthood, if susceptibility to the stresses registered in their teeth also predisposed them to early mortality. Subadults are excluded here to minimize this "mortality selection" bias (Cook, 1981; Goodman and Armelagos, 1988). Age at death was estimated using multiple indicators, including dental development, epiphyseal fusion, ectocranial suture closure, pubic symphysis morphology, auricular surface seriation, cemental annulation, and dental attrition seriation (Wright, 1994). Adults were divided into young (20–34), middle (35–49), and elderly (50+) classes. A few skeletons could not be aged and are considered as adult only. In the total combined skeletal series, the Chi-square test shows no significant difference in the proportion of young, middle, and elderly adults among the three chronological divisions ($\chi^2 = 3.04$, $P = 0.55$). Sex was estimated using dimorphic features of the cranium and pelvis. Cranial and postcranial metric data from well-preserved skeletons that could be securely sexed on morphological grounds were used to create population-specific discriminant functions to classify the sex of more fragmentary skeletons, although a number of skeletons could not be identified by sex (Wright, 1994). The Chi-square test shows that males, females, and unsexed adult skeletons are equally distributed among the chronological divisions ($\chi^2 = 5.61$, $P = 0.23$). Accordingly, mortality selection and sampling bias with respect to sex are unlikely to contribute to differential patterning in hypoplasias over time.

Dental analysis

Hypoplasias were scored on all teeth recovered from 160 adult skeletons in Pasión burials. Sample sizes for each tooth are smaller due to antemortem tooth loss and excavation/curation loss. In addition, teeth for which a continuous section of labial enamel from the cemento-enamel junction (CEJ) to the cusp could not be observed are excluded. Hypoplasias were scored on the labial surface of each tooth under natural light without magnification. Defect location relative to the CEJ was measured to the

nearest 0.02 mm using sliding calipers at the midpoint of the labial aspect. The labial crown height was also measured in order to control for attrition. Defects were scored as linear enamel hypoplasias (LEH), major growth arrests (MGA), shallow broad depressed zones, or pitted enamel. The overwhelming majority of defects are LEH, so all defects are treated as a single class in the statistical analysis. When both sides were present, the antimere with clearest hypoplastic expression or, in cases of disparate attrition, the antimere with greatest crown height is employed.

The age of each hypoplastic insult was calculated using regression equations that incorporate the crown height of unworn Pasión teeth (Table 1) (Goodman and Rose, 1990; Hodges and Wilkinson, 1990). As Whittington (1992) also found at Copán, Pasión Maya teeth are larger than in the Swedish series from which the formulae of Goodman and Rose (1990) were derived, so a population-specific method is necessary. In formulating the equations, the first year of enamel development was considered to be buried under cuspal enamel in each tooth and therefore not observable (Skinner and Goodman, 1992). I acknowledge that this may be excessive for some teeth, but, because intertooth variation in buried enamel has yet to be quantified, a constant increment is the safest assumption. The ages of enamel formation employed by Goodman (Goodman et al., 1980; Goodman and Rose, 1990) from the Massler et al. (1941) standards are used for most teeth. But I considered mandibular canine amelogenesis to have been complete by 4.5 years, which appears to be a more accurate estimate for Amerindians (Anderson et al., 1976; Fanning and Brown, 1971; Skinner and Goodman, 1992; Ubelaker, 1978) and results in better agreement between the age distributions of I¹ and C₁ defects (Norr, 1991). Using these parameters, the linear regression equations in Table 1 were calculated assuming a constant rate of enamel growth, as advocated by Goodman and Rose (1990).

For each tooth, only hypoplasias observed on a section of enamel that is scorable for all teeth of that type are considered. Cuspal enamel that cannot be scored for all individuals is disregarded in order to control for

TABLE 1. Regression equations used to estimate age of hypoplasia formation¹

Tooth	Unworn crown height			Developmental age		Regression equations ²	Minimum scorable age limit
	Mean	s.d.	N	At cusp	At CEJ		
Maxillary							
I ¹	11.14	0.64	8	1.0	4.5	Age = -0.307x + 4.5	2.5
I ²	10.49	0.82	13	2.0	4.5	Age = -0.238x + 4.5	3.0
C ¹	11.18	1.34	17	1.0	6.0	Age = -0.313x + 6.0	3.5
P ³	8.40	0.78	17	3.0	6.0	Age = -0.357x + 6.0	4.0
P ⁴	7.39	0.71	16	3.5	6.0	Age = -0.338x + 6.0	4.0
M ¹	7.32	0.78	8	1.0	3.5	Age = -0.342x + 3.5	2.0
M ²	7.29	0.56	12	4.0	7.5	Age = -0.480x + 7.5	5.0
Mandibular							
I ₁	9.86	0.89	10	1.0	4.0	Age = -0.304x + 4.0	2.5
I ₂	9.74	0.77	17	1.0	4.0	Age = -0.308x + 4.0	2.5
C ₁	11.61	1.45	11	1.5	4.5	Age = -0.258x + 4.5	2.5
P ₃	8.28	0.67	20	2.0	6.0	Age = -0.483x + 6.0	3.5
P ₄	7.64	0.56	19	3.0	7.0	Age = -0.524x + 7.0	4.5
M ₁	7.69	0.73	9	1.0	3.5	Age = -0.325x + 3.5	2.0
M ₂	7.5	0.76	8	4.0	7.0	Age = -0.400x + 7.0	5.5

¹ All measurements are taken in millimeters and ages calculated in years.

² Where x equals the distance of the hypoplasia from the cemento-enamel junction.

attrition. This procedure further limits the narrow age span for which each tooth records stress events, but it excludes very few hypoplasias because cuspal defects are rare. The presence or absence of hypoplasias within this scorable section is the basic unit of analysis for chronological comparisons. For each tooth, the Chi-square test is used to examine the proportion of individuals affected by hypoplasia between periods. The mean age of hypoplastic stress is also examined for each tooth position. Student's t-test is used to evaluate differences in the mean developmental age of defects between periods.

The Pasión region skeletal series is not an ideal sample for paleoepidemiological research because of its small size and the fact that many skeletons are missing teeth due to both antemortem and postmortem loss. However, it is the only available series from this area of Lowland Guatemala. For any given tooth, the sample constitutes a different subset of the total skeletal series (generally less than 70% of the total); each tooth can be considered as an independent test of the trends observed. Because of the imperfect nature of the database, I reject the null hypothesis with a lower level of confidence ($P \leq 0.10$) than is customary for anthropological data but draw attention to results that attain a higher level of significance.

RESULTS

Hypoplasias are fairly common on Pasión teeth. Judging from the mandibular canine,

the most frequently affected tooth, at least 59% of individuals suffered one or more stress episodes. Chronological patterns in hypoplasia prevalence are apparent for several teeth (Fig. 2; Table 2). However, no statistical differences are evident for the I¹, I², C¹, or C₁ among series. If childhood health had deteriorated markedly over the span of Pasión occupation, then we would expect these teeth to show an increase in defects, but they do not. This result indicates stability in the total stress load over time.

Instead, several posterior teeth show chronological trends which are statistically significant at $P \leq 0.10$ or better. In contingency tables considering all three time periods vs. the presence or absence of hypoplasia (Table 2), the Chi-square test suggests subtle chronological changes in the proportion of P³, I₂, P₃, P₄, and M₂ affected. Subdividing these tables, it is evident that the shifts occur between Late and Terminal Classic times. No statistical differences were detected between the "Early" period and Late Classic Period. Greater proportions of individuals with hypoplasias in the Terminal Classic are found for P³, I₂, and P₄ with respect to the Late Classic Period. For the P₃ and M₂ the differences are only evident when the full chronological series is considered, suggesting a longer term, gradual trend.

As each tooth records stress events for a specific narrow age span, these intertooth patterns have an age component and signal

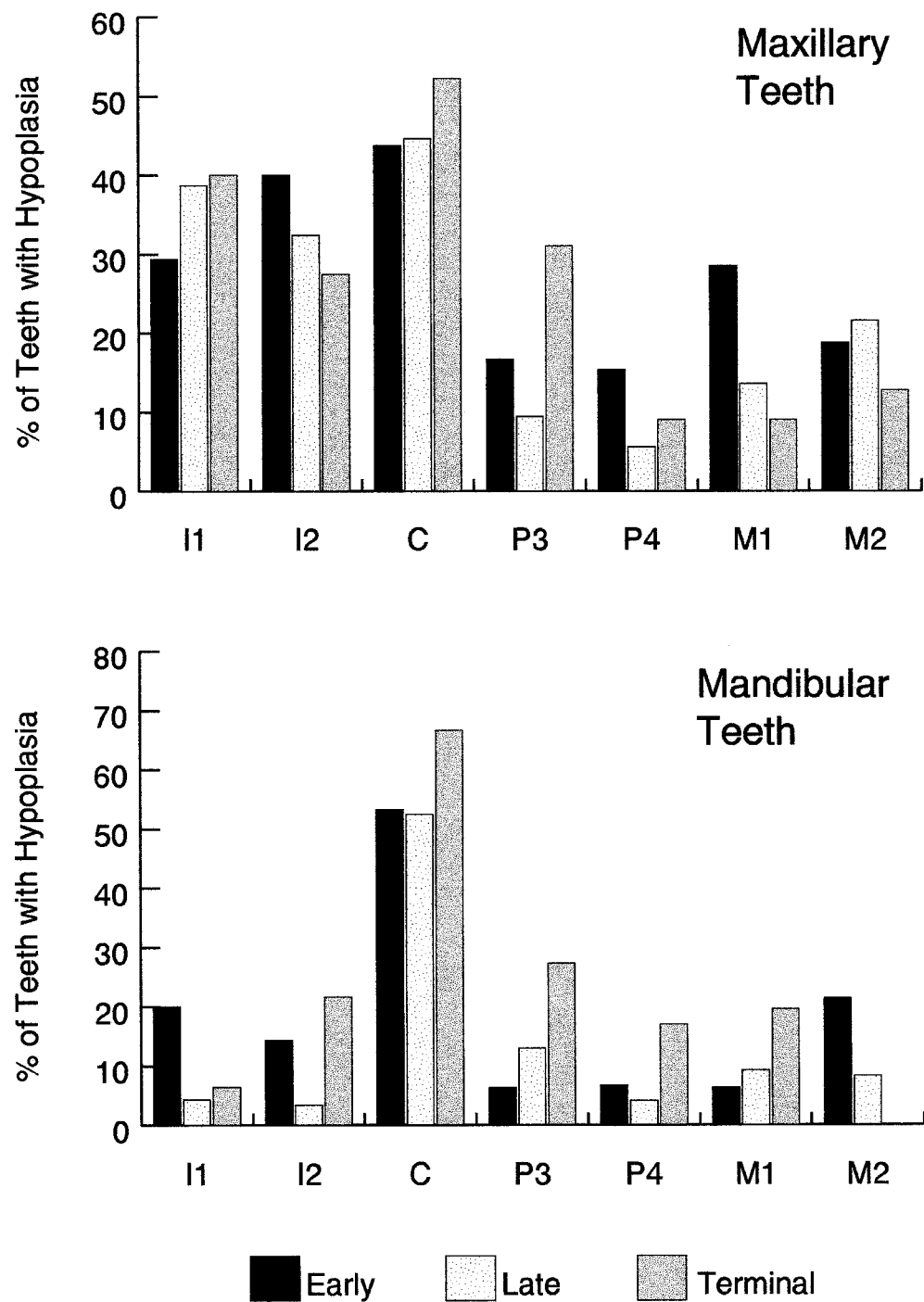


Fig. 2. Histograms showing chronological trends in the proportion of teeth affected by hypoplasias, by tooth position for maxillary teeth (**top**), and mandibular teeth (**bottom**).

TABLE 2. Chi-square test for chronological comparisons of the proportion of teeth affected by hypoplasia by tooth

Tooth	"Early"		Late Classic		Terminal Classic		All 3 periods d.f. = 2		Early-Late d.f. = 1		Late-Terminal d.f. = 1	
	+ ¹	N	+	N	+	N	χ^2	P	$\chi^2_{(Yates)}$	P	$\chi^2_{(Yates)}$	P
Maxillary												
I ¹	5	17	12	31	16	40	0.60	0.74	0.11	0.74	0.02	0.89
I ²	4	10	11	34	11	40	0.64	0.73	0.00	0.94	0.04	0.84
C ¹	7	16	21	47	23	44	0.64	0.73	0.05	0.82	0.26	0.61
P ³	2	12	4	42	14	45	6.39 ²	0.04 ³	0.03	0.86	4.92	0.03 ³
P ⁴	2	13	2	36	4	44	1.20	0.55	0.27	0.60	0.03	0.86
M ¹	4	14	6	44	4	44	3.40	0.18	0.78	0.38	0.11	0.74
M ²	3	16	8	37	6	47	1.19	0.55	0.02	0.89	0.62	0.43
Mandibular												
I ₁	2	10	1	23	2	31	2.52	0.28	0.61	0.44	0.07	0.79
I ₂	2	14	1	29	8	37	4.53	0.10	0.45	0.50	3.15	0.07
C ₁	8	15	21	40	30	45	1.99	0.37	0.06	0.80	1.23	0.27
P ₃	1	16	6	46	12	44	4.84	0.09	0.08	0.78	2.21	0.16
P ₄	1	15	2	49	8	47	4.70	0.09	0.08	0.78	3.03	0.08
M ₁	1	16	4	43	9	46	2.84	0.24	0.02	0.88	1.14	0.28
M ₂	3	14	3	36	0	40	7.92	0.02 ³	0.63	0.43	1.62	0.20

¹ + = number of individuals affected by hypoplasia on a given tooth; N = number of scorable individuals.

² Values of χ^2 in bold are significant at $P \leq 0.10$.

³ Values of χ^2 that also attain significance at $P \leq 0.05$.

a change in the developmental age of hypoplastic stressors over time. The short developmental spans are further restricted here by the minimum age limits that control for variation in attrition (Table 1). For instance, the smaller proportion of M₂ showing hypoplasias over time suggests a decline in stress between the ages of 5 and 7.5 years. The M² absolute numbers of hypoplasias per tooth are also lower in the Terminal Classic, but this is not a statistically significant drop. The M₂ decline hints at slightly better health during older childhood in the final Pasión occupation—at least reduced severity if not frequency of stress. By contrast, hypoplasias occur on significantly more Terminal Classic than Late Classic P³, I₂, and P₄, which record stress between the ages of 4 and 6, 3 and 4.5, and 4.5 and 7 years, respectively. Thus, Terminal Classic children suffered greater occurrence or severity of hypoplastic stressors between the ages of 3 and 6 years than in earlier occupations. Together, these patterns may imply that stressors causing hypoplastic defects shifted toward a younger peak age in the final phase of Pasión Maya history.

Although these patterns do not show extremely high statistical significance, it is important to note that they consistently point to a single trend: a shift in timing of peak stress to a younger age. This agreement is notable in view of the large number

of missing teeth in these poorly preserved skeletons. Because of the imperfect nature of the database, I accept the lower level of confidence ($P \leq 0.10$) in rejecting the null hypothesis that hypoplastic teeth are randomly distributed over time. Note that the proposed shift in the age distribution indicates a subtle change to the chronological pattern of childhood morbidity.

The mean age of hypoplasia was calculated for each tooth and compared across chronological periods using Student's *t*-test (Table 3) to examine this hypothesis further. The paucity of hypoplasias observed on the less sensitive teeth precludes many comparisons. For the maxillary canine, the defects formed at slightly older ages in the Late Classic than they had in the Early Classic Period, albeit by only 3.3 months (0.28 years). However, this shift is not confirmed by any other tooth. By contrast, a significant decrease of 2.8 months (0.23 years) in the mean age of hypoplastic formation was detected for the maxillary canine between the Late and Terminal Classic Periods. This trend is also significant at a higher level of confidence, $P \leq 0.05$. Similarly, mean hypoplastic age is 2.0 months (0.17 years) younger for the Terminal Classic mandibular canine than for the Late Classic canine, also a significant difference. For this tooth the Terminal Classic mean age is also younger than the "Early" period mean age by 2.4

TABLE 3. Student's *t*-test for chronological comparisons of mean age of hypoplasias per tooth

Tooth	“Early” Classic		Late Classic		Terminal Classic		“Early”– Late <i>t</i>	“Early”– Terminal <i>t</i>	Late– Terminal <i>t</i>
	Mean	N	Mean	N	Mean	N			
Maxillary									
I ¹	3.43	6	3.42	13	3.33	21	.06	.56	.64
I ²	3.65	4	3.81	12	3.78	12	—	—	.20
C ¹	4.77	7	5.05	25	4.83	24	–1.45¹	–.26	2.17²
P ³	5.10	2	4.99	8	4.95	14	—	—	.08
P ⁴	5.47	2	5.03	5	5.00	5	—	—	.08
M ¹	2.73	4	2.88	6	2.82	4	—	—	—
M ²	6.63	4	6.07	11	6.22	6	—	—	–.56
Mandibular									
I ₁	3.28	2	3.34	1	3.16	3	—	—	—
I ₂	3.06	2	3.45	1	3.02	11	—	—	—
C ₁	3.51	11	3.48	26	3.31	34	.17	1.45	1.73²
P ₃	4.67	1	4.81	8	4.56	13	—	—	.86
P ₄	6.04	1	6.23	3	5.43	10	—	—	1.85²
M ₁	2.60	1	2.71	4	2.81	8	—	—	—
M ₂	6.42	3	6.31	3	No hypoplasias		—	—	—

¹ Values of *t* in bold are significant at $P \leq 0.10$ for a one-tailed test.² Values also significant at $P \leq 0.05$ for a one-tailed test.

months. Note also that the mean age of arrest is younger for Terminal Classic P₄ than Late Classic ones. Although the P₄ sample is very small, this age shift of 9.6 months is statistically significant. These results provide solid support for the inter-tooth pattern of hypoplasia abundance described above.

DISCUSSION

The distribution of hypoplastic defects within Pasión dentitions parallels that found by Goodman and Armelagos (1985a,b) and supports the hypothesis that teeth differ in sensitivity to ameloblastic disruption. Hypoplasias are most common on the anterior teeth, argued to be the most susceptible to stress. Goodman and Rose (1990) propose that hypoplasias form when genetic, nutritional, and disease factors act to raise ameloblastic disruption above a threshold value. If, however, teeth differ in susceptibility to stress, then genetic and intertooth susceptibility factors would be better viewed as acting on the threshold for a given tooth. Thus, sensitive anterior teeth have lower thresholds than do posterior teeth, so only stressors of greater magnitude are sufficient to reach high hypoplastic thresholds on posterior teeth, while lesser stressors are regularly recorded on anterior teeth. Surveys of dental health that focus on the anterior dentition measure the general incidence and

distribution of stress episodes across childhood. By studying posterior teeth, greater insight is gained into variations in the timing and magnitude of stress within the larger picture.

In the present case, the overall frequency of stress events is shown to have been fairly constant over time, but the relative timing of more severe stress shifted over the span of Pasión history. These results indicate no change in the total stress load suffered by Pasión children over time but rather hint at a decrease in the span of peak childhood illness. The growth of large Late Classic populations may have been associated with a shift to slightly older stress for Late Classic children than that felt by "Early" period children. More pronounced is a Terminal Classic Period shift to stress concentrated at younger ages.

This result conflicts with models of health stress that are commonly invoked to explain the Maya collapse. Ecological models predict a deterioration of childhood health over the Classic Period and that this stress persisted through the Terminal Classic, directly causing site abandonment. These theories have specific implications for childhood health, arguing for increasing reliance on maize as the staple and weaning food (Santley et al., 1986; Willey and Shimkin, 1973). As stated, this should be reflected in increasing total prevalence of hypoplasia on teeth most sus-

ceptible to them over time, particularly in the Terminal Classic Period, a trend that is not substantiated by the data.

The peak age of hypoplastic events is often interpreted as related to the age of weaning in archaeological populations. Peak hypoplasia incidence between ages 2 and 4 is commonly reported in prehistoric skeletal populations, an age not unreasonably old for the termination of breast feeding in such cultures. But, for African-American slave populations, Blakey et al. (1994) found that peak hypoplasia ages are older than historically documented weaning age by 0.5–3.75 years. They suggest that enamel crown susceptibility factors, other environmental stressors, and random factors bias the distribution. Given that most studies emphasize anterior teeth, the role of crown geometry in determining this peak age should not be underestimated (Blakey et al., 1994; Goodman and Armelagos, 1985a,b). As revealed in Table 3, the mean age of hypoplasia differs for each tooth position, in accord with both the developmental span of the tooth and bias from enamel architecture within that span. Although we cannot pinpoint mean age for the total stress experience from these data, the intertooth patterning reveals subtle changes in the timing of stress.

For the Maya, early observations found good agreement between the apparent age of most hypoplasias (3–4 years) and the age of weaning recorded ethnohistorically (Saul, 1972, 1973; Tozzer, 1941). However, some skepticism has been expressed that the Maya could have weaned so late (Danforth, 1989). Today, the mean duration of breast-feeding in Guatemala is 22 months (Pérez-Escamilla, 1993), comparable to that recorded for the Yucatan in the early part of this century (Benedict and Steggerda, 1937). This is among the oldest for national averages in Latin America today (Pérez-Escamilla, 1993), so it seems unlikely that mean weaning age was as late as Bishop Landa testified for the early colonial era (Tozzer, 1941).

As Danforth (1989: 173) notes, weaning is not an abrupt process but begins with very gradual introduction of solid foods, presumably maize gruels (*atole*) in the case of the Maya. By 6 months of age, the growth needs of infants exceed the nutrients supplied in

milk, so that supplementation is necessary. But breast milk is a significant component of the weanling diet for a variable period. It provides an important source of IgA immunoglobulins that build the child's defenses against bacterial and viral intestinal infection (Kleinman and Walker, 1979; Ogra and Losonsky, 1984). For young infants, breast milk provides adequate fluid even in hot climates (Almroth, 1978). For older children, fluid intake from breast milk decreases the amount of water a child drinks. Contaminated water is the primary source of intestinal pathogens that contribute to weanling diarrhea and childhood morbidity. Continued nursing helps to buffer children from illness during this period of dietary and behavioral transition, increasing their chance of survival. Lactation is often prolonged under conditions of food shortage, and low socioeconomic status (SES) children are typically weaned later than those in high SES families (Dow, 1977; Pérez-Escamilla, 1993).

As inferred by Blakey et al. (1994) for American slaves, it is more likely that hypoplasias on Pasi6n teeth represent postweaning stresses (such as infectious agents encountered by ambulatory toddlers) than nutritional inadequacy of the weanling diet alone. Although nutritional deficits do contribute to the abundant hypoplasias of Mesoamerican children today (Goodman et al., 1987; May et al., 1993), the morbidity episodes that may cause enamel defects continue to be frequent after cessation of breast-feeding (Mata and Salas, 1984). Weaning is the most dramatic transition of childhood and almost certainly has some impact on the distribution of dental lesions (Corruccini et al., 1985). It involves a major epidemiological shift as well as the dietary transition.

Although hypoplastic age distributions cannot directly identify the precise age at which lactation was terminated, they do reveal the impact of early child-care practices on the subsequent health of children. The younger age of severe stress among Terminal Classic Pasi6n children observed by hypoplasia patterns on posterior teeth may be due to changes in the population mean duration of lactation. This could have come about either by a shift in weaning age

in the population as a whole or by decreased heterogeneity in weaning age. If mothers systematically withheld breast milk at a younger age, then the timing of postweaning illness should also decline. This would contradict the ecological hypothesis of collapse, in that mothers are expected to prolong lactation under conditions of food stress.

That decreased social heterogeneity in weaning practices might account for the hypoplasia pattern is supported by paleodietary chemistry of adult skeletons from the Pasión sites (Wright, 1994). At Altar de Sacrificios and Seibal, the coefficients of variation of stable carbon isotopes ($\delta^{13}\text{C}$), Sr/Ca, and Ba/Ca ratios are smaller for Terminal Classic skeletons than for their Late Classic ancestors, indicating reduced dietary variability in the Terminal Classic population. Moreover, stable isotopic and trace element signatures of Late Classic skeletons show distinct patterning between burial groups that represent status differences in diet. By contrast, the Terminal Classic mortuary program is less formally structured, and there is no clear chemical patterning between burial groups (Wright, 1994, 1997). Clearly, adult bone chemistry does not correspond directly to childhood diet during amelogenesis. But the chemical data does imply that differential access to foods by adults was decreased during the Terminal Classic Period. If Terminal Classic families had relatively equivalent diets, then child-rearing practices might be more homogeneous than they had been among the more highly stratified Late Classic families. Reduced heterogeneity in child-care practices could produce a greater concentration of stress events around the population central tendency, like the intertooth pattern of hypoplasias documented here.

Evidence for childhood health status from enamel hypoplasias provides little support for hypothesized health deterioration as a causal factor in the Classic Maya collapse. Although dental development does appear to have changed in concert with the sociopolitical transition, it may be more revealing of socioeconomic restructuring than demographic calamity in the final years of Classic Maya society. These results demonstrate that studies of enamel hypoplasia may ben-

efit from observation of the full dentition and from considering tooth positions independently. As crown geometry biases intra-tooth distribution of defects, age trends in hypoplasia prevalence can be monitored by examining tooth positions individually. Although composite scales linking multiple teeth can be used to examine the total distribution of hypoplastic stressors, consideration of individual teeth is one avenue to examine more subtle patterning among stressors of differing magnitude. By focusing on such differences we may be able to gain a more detailed picture of childhood morbidity in prehistory.

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